Pathology of (COVID-19)

Introduction

The first cases of coronavirus disease 2019 (COVID-19) were reported in 31st December 2019, originating in Wuhan, China, with rapid spread worldwide, and COVID-19 became a public health emergency of international concern (WHO, 2020). Up till now the COVID-19 has infected over 1, 174,855 people worldwide and 64,471 deaths, this data is increasing enormously with each passing day date (WHO, 2020). The coronavirus disease 2019 (COVID-19) pneumonia pandemic is caused by a novel coronavirus called severe acute respiratory syndrome 2 (SARS-CoV-2) virus. On the 31st of January 2020, the World Health Organization (WHO) announced that COVID-19 was listed as the Public Health Emergency of International Concern (PHEIC), meaning that it may pose risks to multiple countries and requires a coordinated international response (Li et al., 2020).

It was named COVID-19 by WHO on Feb. 11th, 2020 (Lipsitch et al., 2020). Based on the large number of infected people that were exposed to the wet animal market in Wuhan City, China, it is suggested that this is likely the zoonotic origin of COVID-19. (Rothan and Byrareddy, 2020). These patients were epidemiologically linked to a seafood and wet animal wholesale market in Wuhan, Hubei Province, China. Early reports predicted the onset of a potential Coronavirus outbreak given the estimate of a reproduction number for COVID-19 was deemed to be significantly larger than 1 (ranges from 2.24 to 3.58) (Zhao et al., 2020).

The most common symptoms at onset of COVID-19 illness are fever, cough, and fatigue, while other symptoms include sputum production, headache, haemoptysis, diarrhoea, dyspnoea, and lymphopenia. Clinical features revealed by a chest CT scan presented as pneumonia, however, there were abnormal features such as RNAaemia, acute respiratory distress syndrome, acute cardiac injury, and incidence of grand-glass opacities that led to death (Huang et al., 2020).

The symptoms of COVID-19 infection appear after an incubation period of approximately 5.2 days (Li et al., 2020). The period from the onset of COVID-19 symptoms to death ranged from 6 to 41 days with a median of 14 days. This period is dependent on the age of the patient and status of the patient's immune system. It was shorter among patients > 70-years old compared with those under the age of 70 (Wang et al., 2020).

SARS-CoV-2 is a betacoronavirus. It is comprised of a single-stranded ribonucleic acid (RNA) structure that belongs to the Coronavirinae subfamily, part of the Coronaviridae family. It has a well-defined composition comprising 14 binding residues that directly interact with human angiotensin-converting enzyme 2 (ACE2) (Lu et al., 2020). Importantly, the sequence of the receptor-binding domain of COVID-19 spikes is similar to that of SARS-CoV. This strongly suggests that entry into the host cells is most likely via the ACE2 receptor. (Wan et al., 2020).

SARS-CoV-2 enters the cell via the angiotensin converting enzyme 2 (ACE2) receptor. (Jiang et al., 2020) ACE‐2 is a surface molecule highly expressed in cells of lung, along with esophageal upper epithelial cells and absorptive enterocytes from ileum and colon which indicated digestive system along with respiratory systems is a potential route for SARS‐CoV‐2. (Lu and Shi, 2020) The SARS-Cov-2 first predominantly infects lower airways and binds to ACE2 on alveolar epithelial cells. The virus activates immune cells and induces the secretion of inflammatory cytokines and chemokines into pulmonary vascular endothelial cells. SARS-Cov-2 is a potent inducers of inflammatory cytokines. The “cytokine storm” or “cytokine cascade” is the postulated mechanism for organ damage. (Jiang et al., 2020). Thus, significantly high blood levels of cytokines and chemokines were noted in patients with COVID-19 infection. Patients infected showed higher leukocyte numbers, abnormal respiratory findings, and increased levels of plasma Pro-inflammatory cytokines. Some of the severe cases that were admitted to the intensive care unit showed high levels of pro-inflammatory cytokines including IL2, IL7, IL10, GCSF, IP10, MCP1, MIP1α, and TNFα that are reasoned to promote disease severity (Rothan et al., 2020)

**IL1-β, IL1RA, IL7, IL8, IL9, IL10, basic FGF2, GCSF, GMCSF, IFNγ, IP10, MCP1, MIP1α, MIP1β, PDGFB, TNFα, and VEGFA**.

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